WEST

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L14: Entry 1 of 10 File: DWPI Jul 19, 2001

DERWENT-ACC-NO: 2001-451849
DERWENT-WEEK: 200148

TITLE: Culturing primate pluripotent stem cells for use in therapy, involves culturing in growth environment free of feeder cells but containing conditioned medium produced by collecting medium from feeder cell culture

INVENTOR: CARPENTER, M K; FUNK, W D ; GOLD, J D ; INOKUMA, M S ; XU, C

PATENT-ASSIGNEE: GERON CORP (GERON)

PRIORITY-DATA: 2000US-0688031 (October 10, 2000), 2000US-175581P (January 11, 2000), 2000US-213739P (June 22, 2000), 2000US-213740P (June 22, 2000), 2000US-216387P (July 7, 2000), 2000US-220064P (July 21, 2000)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
WO 200151616 A2	July 19, 2001	E	079	C12N005/06
AU 200111128 A	July 12, 2001		000	C12N005/06
AU 200126395 A	July 24, 2001		000	C12N005/06

DESIGNATED-STATES: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW

APPLICATION-DATA:

PUB-NO	APPL-DATE	APPL-NO	DESCRIPTOR
WO 200151616A2	January 10, 2001	2001WO-US01030	
AU 200111128A	January 11, 2001	2001AU-0011128	
AU 200126395A	January 10, 2001	2001AU-0026395	
AU 200126395A		WO 200151616	Based on

INT-CL (IPC): C07H 21/00; C07K 16/18; C12N 5/06; C12N 5/08; C12N 5/10; C12N 5/22; C12N 15/10; C12P 19/34; C12P 21/02; G01N 33/50

ABSTRACTED-PUB-NO: WO 200151616A
BASIC-ABSTRACT:

NOVELTY - Culturing primate pluripotent stem (pPS) cells, involves culturing in a growth environment essentially free of feeder cells but containing conditioned medium produced by collecting medium from a culture of feeder cells.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a composition (I) comprising pPS cells, which is essentially free of feeder cells;

(2) producing a conditioned medium suitable for culturing pPS cells in a growth environment essentially free of feeder cells, comprising conditioning medium, by culturing cells in the medium, where the cells are euploid cell line that can

proliferate in culture for at least 60 days, and harvesting the conditioned medium;

(3) a conditioned medium (II) to support culturing pPS cells in a growth environment essentially free of feeder cells, produced by the above said method;

(4) a human cell line obtained by differentiating a culture of human embryonic stem (hES) cells into a population of differentiated cells that comprises fibroblast-like cells, and then selecting fibroblast-like cells from the culture, where the conditioned medium produced by harvesting medium from a culture of the fibroblast-like cells supports growth of pPS cells in a culture environment essentially free of feeder cells;

(5) producing (P1) a differentiated cell population, by causing or permitting cells of (I) to differentiate;

(6) producing (P2) differentiated cells from a donor culture of undifferentiated pPS cells, by preparing a suspension of cells from the undifferentiated donor culture, replating and culturing the suspended cells on a solid surface so that they differentiate without forming embryoid bodies, and harvesting differentiated cells from the solid surface;

(7) producing (P3) differentiated cells from a donor culture of pPS cells, by providing a culture of pPS cells that is essentially free of feeder cells, changing the medium in which the cells are cultured, and harvesting differentiated cells after culturing for a time in the changed medium;

(8) a differentiated cell population produced by the above said method;

(9) screening a compound for cellular toxicity or modulation, by contacting a differentiated cell with the compound, determining any phenotypic or metabolic changes in the cell that result from contact with the compound, and correlating the change with cellular toxicity or modulation;

(10) producing a polynucleotide (III) comprising a nucleotide sequence contained in mRNA that is expressed at a different level in committed or differentiated cells compared with undifferentiated pPS cells, by determining the level of expression of a number of mRNAs in committed or differentiated cells, in comparison to the level of expression of the same mRNAs in undifferentiated pPS cells, identifying mRNA expressed at a different level in the committed or differentiated cells, relative to the undifferentiated pPS cells, and preparing a polynucleotide comprising a nucleotide sequence of at least 30 consecutive nucleotides contained in the identified mRNA;

(11) producing genetically altered pPS cells, by:

(a) providing (I), transferring a polynucleotide into pPS cells in (I), and then optionally, selecting the cells that have been genetically altered with the polynucleotide; or

(b) providing a composition of pPS cells on a layer of feeder cells that are drug-resistant, transferring a polynucleotide into pPS cells in the composition, and selecting genetically altered cells in the composition using the drug to which the feeder cells are resistant;

(12) a population of pPS cells, in which at least 25% of the undifferentiated pPS cells have been stably transfected with a polynucleotide, or the progeny of such cells that have inherited the polynucleotide;

(13) a population of genetically altered differentiated cells, obtained by differentiating the above said population of pPS cells;

(14) producing (P4) an mRNA preparation or a cDNA library from pPS cells before or after differentiation, by providing a culture of undifferentiated pPS cells essentially free of feeder cells, optionally permitting the pPS cells to differentiate, and isolating mRNA from the undifferentiated or differentiated cells;

(15) a cDNA library produced by the above said method;

(16) a cDNA library of at least 1000 genes expressed at the mRNA level in either undifferentiated pPS cells, or cells differentiated from pPS cells, where the library is essentially free of cDNA of other vertebrates;

(17) producing a polynucleotide containing a sequence of mRNA expressed in undifferentiated or differentiated pPS cells, by determining nucleotide sequence from mRNA or cDNA obtained by the above said method, and manufacturing a polynucleotide containing the determined sequence;

(18) producing an amino acid containing a sequence of a polypeptide expressed in undifferentiated or differentiated pPS cells, by determining amino acid sequence from a protein encoding region of mRNA or cDNA obtained by the above said method and manufacturing a protein containing the determined sequence; and

(19) producing an antibody specific for a polypeptide expressed in undifferentiated or differentiated pPS cells, by determining amino acid sequence from a protein encoding region of mRNA or cDNA obtained by the above said method, and immunizing an animal or contacting an immunocompetent cell or particle with a protein containing the determined sequence.

ACTIVITY - None given.

MECHANISM OF ACTION - None given.

USE - The method is useful for bulk proliferation of pPS cells for use in studying the biology of pPS cell differentiation and production of important products for use in human therapy. (I) is useful for producing genetically altered pPS cells. Differentiated pPS cells are useful for screening compounds for cellular toxicity or modulation, pharmaceuticals, drugs and toxins. pPS cells are useful for preparing antibody that is specific for embryo markers, stem cell markers, germ cell markers and other antigens expressed on the cells. The pPS cells can be used for tissue re-constitution or regeneration in human patient.

ADVANTAGE - Subtraction libraries with enriched full length cDNA of gene modulated during development can also be obtained. Genetic transfection of feeder-free culture facilitates selection of transfected cells by drug-resistance markers, and gives much higher levels of transient expression. pPS cells can be genetically altered without causing them to differentiate, either on drug-resistant feeders or in feeder-free culture. The feeder-free system has the additional advantage of improving the efficacy of transcription.

ABSTRACTED-PUB-NO: WO 200151616A
EQUIVALENT-ABSTRACTS:

CHOSEN-DRAWING: Dwg.0/15

DERWENT-CLASS: B04 D16 S03
CPI-CODES: B04-F01; B04-F0100E; B11-C09; B12-K04; B12-M05; D05-H08; D05-H09; D05-H14B2; D05-H16A;
EPI-CODES: S03-E14H;

NCBI Nucleotide

PubMed Nucleotide Protein Genome Structure PopSet Taxonomy OMIM Boo

Search Nucleotide for Go Clear

Limits Preview/Index History Clipboard Details

Display default Save Text Add to Clipboard

1: NM_004219. Homo sapiens pitu...[gi:11038651]

Related Sequences, OMIM, Protein, PubMed, Taxonomy, UniSTS, LinkOut

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REFERENCE   1  (bases 1 to 728)
  AUTHORS   Kakar,S.S.
  TITLE     Assignment of the human tumor transforming gene TUTR1 to chromosome
            band 5q35.1 by fluorescence in situ hybridization
  JOURNAL   Cytogenet. Cell Genet. 83 (1-2), 93-95 (1998)
  MEDLINE   99126342
   PUBMED   9925941
REFERENCE   2  (bases 1 to 728)
  AUTHORS   Dominguez,A., Ramos-Morales,F., Romero,F., Rios,R.M., Dreyfus,F.,
            Tortolero,M. and Pintor-Toro,J.A.
  TITLE     hpttg, a human homologue of rat pttg, is overexpressed in
            hematopoietic neoplasms. Evidence for a transcriptional activation
            function of hPTTG
  JOURNAL   Oncogene 17 (17), 2187-2193 (1998)
  MEDLINE   99027589
   PUBMED   9811450
REFERENCE   3  (bases 1 to 728)
  AUTHORS   Zhang,X., Horwitz,G.A., Heaney,A.P., Nakashima,M., Prezant,T.R.,
            Bronstein,M.D. and Melmed,S.
  TITLE     Pituitary tumor transforming gene (PTTG) expression in pituitary
            adenomas
  JOURNAL   J. Clin. Endocrinol. Metab. 84 (2), 761-767 (1999)
  MEDLINE   99145048
   PUBMED   10022450
REFERENCE   4  (bases 1 to 728)
  AUTHORS   Kakar,S.S. and Jennes,L.
  TITLE     Molecular cloning and characterization of the tumor transforming
            gene (TUTR1): a novel gene in human tumorigenesis
  JOURNAL   Cytogenet. Cell Genet. 84 (3-4), 211-216 (1999)
  MEDLINE   99321714
   PUBMED   10393434
REFERENCE   5  (bases 1 to 728)
  AUTHORS   Pei,L.
  TITLE     Pituitary tumor-transforming gene protein associates with ribosomal
            protein S10 and a novel human homologue of DnaJ in testicular cells
  JOURNAL   J. Biol. Chem. 274 (5), 3151-3158 (1999)
  MEDLINE   99115663
   PUBMED   9915854
REFERENCE   6  (bases 1 to 728)
  AUTHORS   Zhang,X., Horwitz,G.A., Prezant,T.R., Valentini,A., Nakashima,M.,
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            Bronstein,M.D. and Melmed,S.
  TITLE     Structure, expression, and function of human pituitary
            tumor-transforming gene (PTTG)
  JOURNAL   Mol. Endocrinol. 13 (1), 156-166 (1999)
  MEDLINE   99107039
   PUBMED   9892021
REFERENCE   7  (bases 1 to 728)
  AUTHORS   Zou,H., McGarry,T.J., Bernal,T. and Kirschner,M.W.
  TITLE     Identification of a vertebrate sister-chromatid separation
            inhibitor involved in transformation and tumorigenesis
  JOURNAL   Science 285 (5426), 418-422 (1999)
  MEDLINE   99340303
   PUBMED   10411507
REFERENCE   8  (bases 1 to 728)
  AUTHORS   Kakar,S.S.
  TITLE     Molecular cloning, genomic organization, and identification of the
            promoter for the human pituitary tumor transforming gene (PTTG)
  JOURNAL   Gene 240 (2), 317-324 (1999)
  MEDLINE   20047911
   PUBMED   10580151
COMMENT     REVIEWED REFSEQ: This record has been curated by NCBI staff. The
            reference sequence was derived from AF095287.1.
            On Oct 30, 2000 this sequence version replaced gi:4758979.
            Summary: The encoded protein is a homolog of yeast securin
            proteins, which prevent separins from promoting sister chromatid
            separation. It is an anaphase-promoting complex (APC) substrate
            that associates with the separin ESP1 until activation of the APC.
            The gene product has transforming activity in vitro and tumorigenic
            activity in vivo, and the gene is highly expressed in various
            tumors. The gene product contains 2 PXXP motifs, which are required
            for its transforming and tumorigenic activities, as well as for its
            stimulation of basic fibroblast growth factor expression. It also
            contains a destruction box (D box) that is required for its
            degradation by the APC. The acidic C-terminal region of the encoded
            protein can act as a transactivation domain. The gene product is
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            COMPLETENESS: complete on the 3' end.
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Revised: October 24, 2001.

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Search Results -

Terms	Documents
restor$5 near diploid$1	1

Database:
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JPO Abstracts Database
EPO Abstracts Database
Derwent World Patents Index
IBM Technical Disclosure Bulletins

Refine Search: restor$5 near diploid$1 Clear

Search History

Today's Date: 2/4/2002

DB Name	Query	Hit Count	Set Name
USPT	restor$5 near diploid$1	1	L39
USPT	treat$5 near3 l34	6	L38
USPT	(chemotherap$3 or therap$3) near3 l34	0	L37
USPT	l34 with (eliminat$3 or kill$3)	7	L36
USPT	l34 with target$3	12	L35
USPT	(aneuploid$1 or euploid$1 or polyploid$1)	764	L34
USPT	l32 and @ad<20010323	296	L33
USPT	l31 and (aneuploid$1 or euploid$1 or polyploid$1)	296	L32
USPT	l30 with (treatment$1 or therap$3 or chemotherap$3)	27525	L31
USPT	(tumor$1 or tumour$1 or cancer$1)	62078	L30
USPT	l19 and (chemotherap$3)	2	L29
USPT	l19 and (treatment$1 or therap$3)	55	L28
USPT	l26 and spindle	0	L27

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USPT	l19 with (tumor$1 or tumour$1 or cancer$1)	0	L23
USPT	l19 same centrosome$1	0	L22
USPT	l19 same spindle$1	0	L21
USPT	l19 with spindle$1	0	L20
USPT	(aneupolid$1 or euploid$1)	57	L19
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JPAB,EPAB,DWPI	l14 with centrosome$1	0	L17
JPAB,EPAB,DWPI	l14 same spindle	0	L16
JPAB,EPAB,DWPI	l14 with spindle	0	L15
JPAB,EPAB,DWPI	(aneupolid$1 or euploid$1)	10	L14
JPAB,EPAB,DWPI	(h-securin$1) or (h-PTTG$1)	0	L13
JPAB,EPAB,DWPI	l11 and human	3	L12
JPAB,EPAB,DWPI	pds1 or cut2	11	L11
JPAB,EPAB,DWPI	hpttg$1 or vsecurin$1 or hsecurin$1	0	L10
JPAB,EPAB,DWPI	l8 near human	24	L9
JPAB,EPAB,DWPI	securin$1 or pttg$1 or (pituitary adj2 transform$5 adj (gene$1 or protein$1 or peptide$1 or polypeptide$1))	144943	L8
USPT	(h-securin$1) or (h-PTTG$1)	0	L7
USPT	l4 with (human near homolo$4)	0	L6
USPT	l4 with (human adj homolo$4)	0	L5
USPT	pds1 or cut2	78	L4
USPT	hpttg$1 or vsecurin$1 or hsecurin$1	0	L3
USPT	human adj l1	4	L2
USPT	securin$1 or pttg$1 or (pituitary adj2 transform$5 adj (gene$1 or protein$1 or peptide$1 or polypeptide$1))	284151	L1

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End of Result Set

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L26: Entry 1 of 1 File: USPT Dec 21, 1999

DOCUMENT-IDENTIFIER: US 6004528 A
TITLE: Methods of cancer diagnosis and therapy targeted against the cancer stemline

DEPR:
By the OSES model then the phenomenon of tumor "progression" would correspond to a gross histopathologic change due to the gradual emergence of pre-existing cancer cells rather than the stepwise selection of individual "pre-cancerous" intermediates. The preferential outgrowth of a clone(s) of cancer cells from amongst its slower-growing (i.e. differentiating/regressing) neighbors could then be attributed to the advantageous acquisition of mutations which inhibit that mutant clone's ability to differentiate. More specifically, by the OSES model peripherally-located cancer cells in a tumor mass may be the most susceptible population to selective pressure for differentiation-impairing mutations thereby effectively "shielding" the more centrally-located cancer cells of such pressures (and thus of significant mutational accumulation). Accordingly, expansion of peripherally-located mutants which are differentiation-defective (and which may or may not have a limited proliferative capability) might further shield a more centrally-located subpopulation thereby allowing it to expand and act as an "immortal" founder line (i.e. cancer stemline) that is relatively mutationally-spared. This idea is consistent with reports that certain human tumors (e.g. breast carcinomas) have a higher histopathological grade in their more central regions as well as with findings that some highly aneuploid tumor types have a chromosomal distribution pattern best accounted for by the presence of a stemline which is neareuploid (Lennington et al, "Ductal carcinoma in situ of the breast. Heterogeneity of individual lesions", Cancer, 73:118-124 (1994); Makino, "Further Evidence Favoring The Concept of the Stem Cell In Ascites Tumors Of Rats", Ann. N.Y. Acad. Sci., 63:818-830 (1956); Shapiro et al, "Isolation, Karyotype, and Clonal Growth of Heterogeneous Subpoplations of Human Malignant Gliomas", Cancer Res., 41:2349-2357 (1981)). If some mutations could affect tumor growth behavior subsequent to the birth of a cancer cell (e.g. by inhibiting cancer cell differentiation) then another potential non-neo-Darwinian explanation for an inherited cancer predisposition (in addition to predisposing to the early "initiation" stage of cancer by causing non-stem cells to aberrantly differentiate) might also derive from germline inheritance of a mutant gene that accelerates the latter "progression" stage of cancer by thwarting differentiation/reversion of cells which are already cancerous.

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L36: Entry 1 of 7 File: USPT Jun 12, 2001

DOCUMENT-IDENTIFIER: US 6245501 B1
TITLE: Method for cancer screening

DEPR:
By way of hypothesis but not limitation, it appeared that in aneuploid cells (A10 and ACHN) endoapoptosis was caused by karyotype (genotype) instability, resulting in the elimination of the defective daughter cells. In irradiated tumor cells, DNA strand breaks initiate multinucleate endoapoptotic cells. Then, it follows that in preneoplastic cells such as NIH3T3 and A10 cells, prolonged cell--cell contact under confluence may induce increased karyotype (genotype) instability and therefore, endoapoptosis.

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L38: Entry 3 of 6 File: USPT Nov 22, 1994

DOCUMENT-IDENTIFIER: US 5366885 A
TITLE: Method and kit for testing tumors for drug sensitivity

DEPR:
In FIG. 9, the results of two different doses of Adriamycin (0.5 or 5 .mu.g/ml for 24 hours) on an adenocarcinoma of the colon are shown. In the diploid cohort of cells, the lowest Adria dose caused an S-phase arrest; the higher Adria dose caused a G.sub.1 phase arrest. Bleo also caused a G.sub.1 arrest. In the aneuploid segment of this tumor, the lower Adria dose had no effect (compared to untreated controls), while the higher Adria dose still produced a G.sub.1 blockade. Bleo caused a large S-phase block in these aneuploid cells. G.sub.2 M phase blockades occurred with both Adria doses in diploid cells. Diploid cells exposed to Bleo showed a large decrease in G.sub.2 M phase cells, probably the result of the G.sub.1 blockade. Aneuploid cells treated with both Adria doses were not affected. A slight G.sub.2 M phase block was observed after Bleo treatment. Since GSH values were in the normal range, no interference by GSH on drug effects were expected or observed.

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L14: Entry 5 of 10 File: DWPI May 9, 2000

DERWENT-ACC-NO: 1997-434734
DERWENT-WEEK: 200032

TITLE: Cytokine, designated ESRF, used to establish and propagate embryonic stem cells - also new cell lines producing the cytokine

INVENTOR: BEUHR, M L; CHAMBERS, I P ; DANI, C ; SMITH, A G ; BUEHR, M L

PATENT-ASSIGNEE: UNIV EDINBURGH (UYEDN)

PRIORITY-DATA: 1996GB-0011319 (May 31, 1996), 1996GB-0003244 (February 16, 1996)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
JP 2000505294 W	May 9, 2000		052	C12N015/09
WO 9730151 A1	August 21, 1997	E	058	C12N015/01
AU 9718027 A	September 2, 1997		000	C12N015/01
ZA 9701302 A	November 26, 1997		054	A61K000/00
EP 880584 A1	December 2, 1998	E	000	C12N015/01

DESIGNATED-STATES: AL AM AT AU AZ BB BG BR BY CA CH CN CZ DE DK EE ES FI GB GE HU IL IS JP KE KG KP KR KZ LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TR TT UA UG US UZ VN AT BE CH DE DK EA ES FI FR GB GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG AT BE CH DE DK ES FI FR GB GR IE IT LI LT LU LV MC NL PT SE SI

CITED-DOCUMENTS:5.Jnl.Ref; JP 05304951 ; JP 07051060 ; JP 08154681 ; WO 9002183

APPLICATION-DATA:

PUB-NO	APPL-DATE	APPL-NO	DESCRIPTOR
JP2000505294W	February 14, 1997	1997JP-0529116	
JP2000505294W	February 14, 1997	1997WO-GB00426	
JP2000505294W		WO 9730151	Based on
WO 9730151A1	February 14, 1997	1997WO-GB00426	
AU 9718027A	February 14, 1997	1997AU-0018027	
AU 9718027A		WO 9730151	Based on
ZA 9701302A	February 17, 1997	1997ZA-0001302	
EP 880584A1	February 14, 1997	1997EP-0903477	
EP 880584A1	February 14, 1997	1997WO-GB00426	
EP 880584A1		WO 9730151	Based on

INT-CL (IPC): A61K 0/00; A61K 38/00; A61P 43/00; C07K 14/52; C12N 5/10; C12N 15/01; C12N 15/09; C12N 5/10; C12N 15/09; C12R 1/91; C12R 1/91

ABSTRACTED-PUB-NO: WO 9730151A
BASIC-ABSTRACT:

A novel cytokine designated ESRF is characterised by the capacity to inhibit differentiation of embryonic stem (ES) cells (i) in the absence of DIA/LIF (differentiation inhibiting activity/myeloid leukaemia inhibitory factor; (ii) in

the absence of cytokines which act through gp 130, and (iii) in the absence of interaction with gp130. Also claimed are: (1) a cell line, designated D7A3-PE (ECCAC94111845), and (2) an established line of ES cells characterised by possessing at at least 5, preferably at least 7 of the following: (i) the characteristic morphology of stem cells, including growth in clumps as small tightly packed cells with a high nuclear to cytoplasmic ratio; (ii) expression of at least 1 specific marker, selected from alkaline phosphatase, stage-specific embryonic antigen, and Oct-3/4; (iii) non-expression of differentiation markers, e.g. H19 RNA; (iv) substantial or unlimited propagation potential; (v) stability to freezing and thawing; (vi) stable euploid karyotype, preferably other than mouse; (vii) propagation dependent on cytokines; (viii) in vitro differentiation induceable by withdrawal of cytokines, aggregation or chemical differentiation inducers; (ix) ability to form teratocarcinomas comprising derivatives of endoderm, mesoderm, and ectoderm; (x) ability to colonise and/or reconstitute host tissues through the production of somatic stem cells and functionally differentiated progeny; (xi) ability to colonise host embryos with contribution of functionally differentiated progeny to chimeras; (xii) ability to produce functional gametes in chimeras and generation of viable offspring, and (xiii) ability to integrate exogenous DNA.

ESRF is capable of inhibiting differentiation of ES cells in the absence of interaction with LIF-receptor. Inhibition of differentiation is effected by a mechanism which is distinct from gp130. The ESRF is obtainable by culturing the cell line D7A3-PE. Its capacity to inhibit differentiation cannot be eliminated by neutralising anti-CNTF antiserum, or anti-gp130 antiserum. The cell line of (2) has at least 5 (preferably at least 7) of the features (I)-(viii). The cell line also has at least 1 (preferably at least 2) of the features (ix)-(xiii). The cell line preferably has a demonstrated rat karyotype.

USE - ESRF can be used in establishing ES cells, and in propagating ES cells (claimed). ESRF can be used to isolate and maintain in vitro ES cells from other murine strains, and from other species including laboratory animals (e.g. rats, rabbits and guinea pigs), domesticated animals (e.g. sheep, goats, horses cattle and pigs), and primates, which has not previously been possible.

ABSTRACTED-PUB-NO: WO 9730151A
EQUIVALENT-ABSTRACTS:

CHOSEN-DRAWING: Dwg.0/5

DERWENT-CLASS: B04 C06 D16
CPI-CODES: B04-F01; B04-H01; B14-L01; C04-N02; C14-H01; D05-H08; D05-H09;

WEST

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L14: Entry 6 of 10 File: DWPI Jul 25, 1996

DERWENT-ACC-NO: 1996-354519
DERWENT-WEEK: 200174

TITLE: Purified primate embryonic stem cells capable of long term culture - for producing transgenic primates as models of human disease, and for prepn. of tissue transplants

INVENTOR: THOMSON, J A

PATENT-ASSIGNEE: WISCONSIN ALUMNI RES FOUND (WISC)

PRIORITY-DATA: 1995US-0376327 (January 20, 1995)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
WO 9622362 A1	July 25, 1996	E	050	C12N005/00
AU 9647584 A	August 7, 1996		000	C12N005/00
EP 770125 A1	May 2, 1997	E	000	C12N005/00

DESIGNATED-STATES: AL AM AT AU AZ BB BG BR BY CA CH CN CZ DE DK EE ES FI GB GE HU IS JP KE KG KP KR KZ LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TR TT UA UG UZ VN AT BE CH DE DK EA ES FR GB GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG AT BE CH DE DK ES FR GB GR IE IT LI LU MC NL PT SE

CITED-DOCUMENTS:4.Jnl.Ref

APPLICATION-DATA:

PUB-NO	APPL-DATE	APPL-NO	DESCRIPTOR
WO 9622362A1	January 19, 1996	1996WO-US00596	
AU 9647584A	January 19, 1996	1996AU-0047584	
AU 9647584A		WO 9622362	Based on
EP 770125A1	January 19, 1996	1996EP-0903521	
EP 770125A1	January 19, 1996	1996WO-US00596	
EP 770125A1		WO 9622362	Based on

INT-CL (IPC): C12N 5/00

RELATED-ACC-NO: 1999-094399;2001-256470 ;2001-638504

ABSTRACTED-PUB-NO: WO 9622362A
BASIC-ABSTRACT:

Purified prepn. of primate embryonic stem cells (ESC) has the following properties: (a) can proliferate in vitro for over a year; (b) maintain the normal karyotype, and retain ability to differentiate to derivs. of endoderm, mesoderm and ectoderm, throughout long-term culture; and (c) will not differentiate on a fibroblast feeder layer.

USE - The cells are used to generate transgenic primates as models of specific human genetic diseases where the gene responsible has been cloned, and in tissue transplants by adjusting culture conditions to generate specific cell types

(blood, neurological or muscle cells), or by allowing the cells to differentiate in tumours. The differentiated cells can also be isolated, and transplanted to treat haematopoietic, endocrine or degenerative neurological disease or hair loss, e.g. Parkinson's disease, juvenile onset diabetes or AIDS.

ADVANTAGE - The cells resemble human cells, and can be kept in the undifferentiated state, while remaining euploid, for long periods.

ABSTRACTED-PUB-NO: WO 9622362A
EQUIVALENT-ABSTRACTS:

CHOSEN-DRAWING: Dwg.0/6

DERWENT-CLASS: B04 D16
CPI-CODES: B04-F02; B04-P01A0E; B12-K04A; B14-F02; B14-G01B; B14-H02; B14-J01A3; B14-J01A4; B14-R02; B14-S04; D05-H16A;

WEST

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L35: Entry 9 of 12 File: USPT Dec 9, 1997

DOCUMENT-IDENTIFIER: US 5696153 A
TITLE: Therapeutic regimen for treating patients

DEPR:
Initial experiments were conducted to test the cytotoxic effects of taxol on in vitro cell cultures of cancerous cells. The target indicators measured were cell growth arrest, necrosis, apoptosis, polyploidy and cytophagia. Experimental design included: (1) determination of the dose/time/toxicity relationship following exposures to taxol of 15 minutes to 24 hours in cell lines; (2) determination of the lowest effective dose; and (3) the effect of brief multiple serial challenges with taxol, at intervals of hours to weeks.